INFECTIONS

- The Infected Foot Ulcer
- Soft Tissue Infection Under Callus
- Infection Under Callus over Fifth Toe
- Soft Tissue Infection
- Web Space Infection
- Onychomycosis
- Fungal Infection with Multimicrobial Colonization
- Deep Tissue Infection After Interphalangeal Mycosis
- Deep Tissue Infection
- Deep Tissue Infection of a Charcot Foot with a Neuropathic Ulcer
- Osteomyelitis
- Osteomyelitis of the Hallux
- Phlegmon
- Infected Plantar Ulcer with Osteomyelitis
- Neuropathic Ulcer with Osteomyelitis
- Osteomyelitis of the First Metatarsal Head
- Chronic Neuropathic Ulcer with Osteomyelitis
- Bone Scintigraphy Imaging
- Osteomyelitis of the Heel

THE INFECTED FOOT ULCER

Invasion of foot tissues by microorganisms, usually accompanied by an inflammatory response, may follow colonization of the skin by initially harmless bacteria, or occur as a primary event.

Diagnosing a foot ulcer infection is based on clinical criteria. A superficial or a full-thickness ulcer, treated inadequately, predisposes to infection, although cellulitis or osteomyelitis can occur without a break in the skin. Infected ulcers are often asymptomatic, especially if the patient feels no pain, due to diabetic polyneuropathy; or they may cause mild discomfort, and produce some drainage, which eventually may become purulent and odorous. Disturbance of blood glucose control may be early evidence of a local infection.

Clinical assessment of any ulcer includes description of location, appearance, extent, depth, temperature, and odor. *Appearance* includes color, type and condition of tissue, presence of drainage, an eschar, necrosis or surrounding callus. Infected wounds may be purple or red, or even brown or black, depending on the pathogen and its etiology, and their drainage may be serous, hemorrhagic or purulent. Induration of the skin and swelling usually denotes infection. *Extent* is measured either directly on a clear film, or by defining length and width of the ulcer. *Depth* is estimated with a sterile blunt probe, which also determines underlying sinus tracts, abscesses or penetration to a bone. *Temperature* is estimated by the hand of the examiner or measured by a dermal thermometer. Underlying infections raise skin temperature. Foul *odor* of an ulcer may denote infection by a specific pathogen (such as *Proteus, Pseudomonas*, anaerobes, a mixed infection, or fungi), or, simply a necrotic process.

Wound infections are categorized as mild, moderate or severe. *Mild* infections are superficial infections confined to the skin and the subcutaneous fat, with minimal or no purulence or cellulitis. *Moderate* infections are deep and may involve fasciae, muscles, tendons, joints or bones. They may present as cellulitis of 0–2 cm in diameter, or a plantar abscess, and they may cause systemic symptoms; they impose a certain risk of amputation. *Severe* infection of a foot ulcer is a deep infection with more than 2 cm of cellulitis, lymphangitis, gangrene, and/or necrotizing fasciitis, threatening limb loss and causing systemic toxicity. Absence of symptoms or signs of systemic illness does not exclude a limb-threatening infection.

Cultures for aerobic and anaerobic pathogens and fungi assist in the management of infection. Curettage of the base of the debrided ulcer, culture of material collected by surgical biopsy of deep tissue or bone, or aspiration of drainage are preferred; superficial swab cultures are not usually helpful, since they often produce different results from cultures of deep tissues. They are also difficult to interpret because of the number of pathogens found on the surface of a wound and they are unsuitable for anaerobes.

Before culturing a wound, any overlying necrotic tissue should be removed by vigorous scrubbing with saline-moistened sterile gauze.

Bacteria that colonize normal skin are coagulase-negative staphylococci, α-hemolytic streptococci and other gram-positive aerobes, and corynebacteria. *Staphylococcus aureus* or β-hemolytic streptococci,

pathogens that colonize the skin of diabetic patients, are the causative agents of acute infections in antibiotic-naïve patients, and are nearly always the cause of cellulitis in non-ulcerated skin; *Staphylococcus aureus* is the most commonly recovered pathogen in most infections in which a single agent is isolated. Polymicrobial cultures, with an average of five or six organisms, are often obtained from patients with chronic lesions, especially when they have been treated with antibiotics for some time; anaerobes, mostly *Bacteroides* sp. and various anaerobic gram-positive cocci are often isolated from deep necroses; *Proteus* spp. and *Escherichia coli* predominate among gram-negative bacilli; and *Pseudomonas* is often isolated from indurated, wet wounds. In severe infections, gram-negative pathogens and anaerobes predominate, versus gram-positive pathogens and enterobacteriaceae, which are usually isolated from mild infections. Severity of infection does not predict the causative microorganism.

Colonization of skin with any bacterium without concomitant infection, does not need treatment; therefore culture results should be interpreted with caution.

Mild or moderate cellulitis may be treated with dicloxacillin, first-generation cephalosporins or clindamycin. Cotrimoxazole is an alternative for treating a proven staphylococcal infection, as well as for enterobacteriaceae.

When infection is mild and the causative pathogens and their susceptibility to antibiotics are predictable, empirical antibiotic therapy is justified. A narrower-spectrum agent may be chosen, such as first-generation cephalosporins and/or clindamycin. One to three weeks of therapy may suffice for soft tissue infections. The more severe the infection and the higher the prevalence of antibiotic resistance, the greater the need for microbiological information. Second-generation oral cephalosporins, amoxicillin–clavulanic acid or fluoroquinolones, and clindamycin or metronidazole may be effective oral treatment against moderate infections, for patients who do not need hospitalization, when a mixed infection is suspected or before microbiological data are available.

Patients with severe infections, and those in a systemic toxic condition, should be hospitalized and treated with intravenous antibiotics, since they may be unable to swallow or tolerate oral therapy, in addition to which more predictable levels of antibiotics in infected tissues can be achieved with intravenous administration. Such patients are usually treated with broad-spectrum antibiotics, before cultures and antibiograms are available. Imipenem–cilastin, ampicillin–sulbactam, piperacillin–tazobactam, third-generation cephalosporins or fluoroquinolones are usually effective; clindamycin or metronidazole may be added and an agent with a narrower spectrum may be chosen later based on the antibiograms. Urgent surgical drainage or removal of dead tissues may also be needed. Arterial insufficiency may compromise therapy as it prevents the antibiotics from reaching the site of infection. In this case surgical revascularization of the limb is carried out.

Radiographic evaluation is necessary when osteomyelitis is suspected or for wounds of long duration. Subcutaneous gas, foreign bodies, fractures, cortical erosions or neuro-osteoarthropathy may be seen on plain radiographs.

Keywords: Infection, clinical assessment; infection, diagnosis; cultures, curettage; infection, mild; infection, moderate; infection, severe

SOFT TISSUE INFECTION UNDER CALLUS AT THE TIP OF A CLAW TOE

In the case of claw toe deformity of the second toe, callus formation and onychodystrophy was due to exposure of the tip of the toe to high pressure during the propulsion phase of gait.

A purulent discharge was evident after removal of the callus (Figure 8.1). The patient felt no pain or discomfort due to severe peripheral neuropathy. No other signs of infection were present on the toe or the forefoot, therefore drainage of pus was adequate, and no further treatment was needed. Appropriate footwear was prescribed.

Keywords: Soft tissue infection; claw toe; onychodystrophy; subungual hemorrhage; third ray amputation

INFECTION UNDER CALLUS OVER FIFTH TOE

A 66-year-old female patient, with type 2 diabetes which was diagnosed at the age of 51 years was referred to the diabetic foot clinic for chiropody treatment. On physical examination she had severe peripheral neuropathy, and bounding pedal pulses. Superficial vein dilatation on the dorsal foot, dry skin — as a result of neuropathy — and hyperkeratosis over the fifth metatarsal head were noted. Painless hyperkeratosis was seen over the dorsal aspect of the left little toe, caused by inappropriate footwear. After callus removal a purulent discharge was noticed (Figure 8.2). A coagulase-negative *Staphylococcus aureus* was isolated from the pus. A plain radiograph was negative for bone involvement. No antibiotic was given since the pus had drained completely. The ulcer healed within 2 weeks after local treatment.

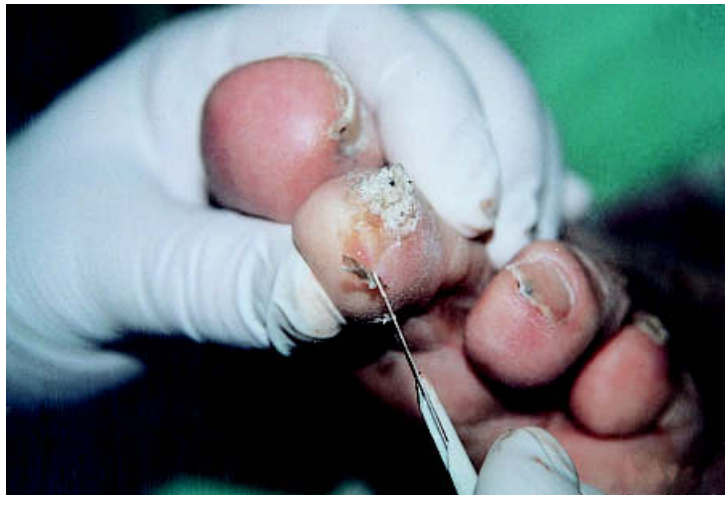

Figure 8.1 Purulent discharge was evident after removal of callus from the tip of this second claw toe with onychodystrophy. A third ray amputation was carried out 2 years ago. A subungual hemorrhage due to intense trimming of the nail of the fourth toe can be seen

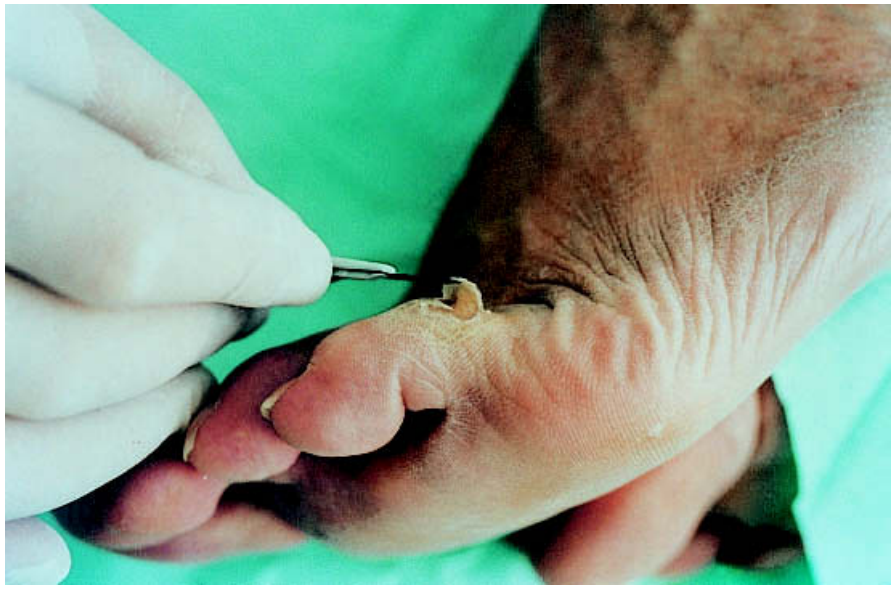

Figure 8.2 Superficial vein dilatation can be seen on the dorsal foot along with dry skin and hyperkeratosis over the fifth metatarsal head. There was painless hyperkeratosis over the dorsal aspect of the left little toe. After callus removal purulent discharge was noted

Non-limb-threatening infections are usually caused by gram-positive cocci, typically *Staphylococcus aureus* and *Streptococcus* spp. In hospitalized patients with diabetic foot infections, methicillin-resistant *Staphylococcus aureus* as well as enterococci are more prevalent.

Keywords: Infection; callus, claw toes

SOFT TISSUE INFECTION

A 58-year-old male patient with type 2 diabetes, diagnosed at the age of 45 years, attended the outpatient diabetic foot clinic for chiropody treatment on a fortnightly basis. He had severe peripheral neuropathy, claw toe deformity and prominent metatarsal heads. He mentioned mild pain on his left midsole on finding a coin in his shoe after prolonged walking.

On examination, he had a lax blister containing purulent fluid under his left midsole. There was extensive surrounding erythema and callus formation under the second metatarsal head (Figure 8.3). The blister was removed and dressed, and the patient was advised to use crutches and walk on his heel. He was treated with clindamycin for 2 weeks. Debridement of the callus was also carried out.

The blister had developed following an unrecognized trauma to the foot. Such injuries are detrimental to patients with loss of sensation. The coin in the patient's shoe put an additional load under his midsole. All patients with loss of protective sensation should be instructed to inspect and feel the inside of their shoes before they wear them. A selection of objects collected from

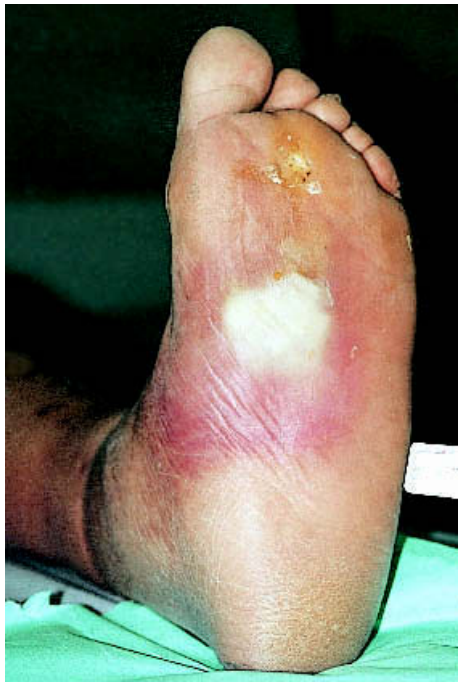

Figure 8.3 A lax blister containing purulent fluid under the midsole with extensive surrounding erythema. Injury caused by a coin in the shoe which was not felt by the patient. Callus formation under the second metatarsal head is apparent

patients' shoes at the outpatient diabetic foot clinic is shown in Figure 8.4.

Diabetic bullae may also cause blisters in diabetic patients. They occur on the lower legs, the dorsum of the feet, hands, and forearms and less commonly, under the soles of the feet. Diabetic bullae more often affect men. They appear suddenly as tense and usually bilateral blisters, with diameters of 0.5 to several cm; they contain clear fluid without any surrounding erythema and heal in a few weeks without scarring. Relapses are common.

Keywords: Trauma; in-shoe foreign objects; diabetic bullae

WEB SPACE INFECTION

A 72-year-old female patient with type 2 diabetes diagnosed at the age of 61 years, was referred to the outpatient diabetic foot clinic because of an infection in her left foot. The patient had poor diabetes control (HBA_{1c}: 8.5%), and was being treated with insulin twice daily. She had background

Figure 8.4 Objects collected from patients' shoes. Loss of protective sensation prevents patients from feeling injurious stimuli

diabetic retinopathy and diabetic nephropathy. She reported itching in the fourth interdigital space 6 months previously, probably due to a fungal infection.

On examination, peripheral pulses were weak and she had severe loss of sensation. There was significant ankle edema. A full-thickness painless, infected neuro-ischemic ulcer was present in the fourth web space, with exposure of the subcutaneous tissue (Figure 8.5). No signs of systemic toxicity were found. The ankle brachial index was 0.7. The ulcer was debrided and dressed. Swab cultures obtained from the base of the ulcer revealed *Staphylococcus aureus* and *Escherichia coli*. A plain radiograph excluded osteomyelitis. She was treated with amoxicillin–clavulanic acid for 2 weeks. Oral furosemide was initiated in order to reduce the ankle edema. The patient had her wound dressed at home daily and attended the outpatient diabetic foot clinic on a weekly basis. A triplex examination of the leg arteries revealed the presence of moderate stenosis in the left superficial femoral and left popliteal arteries. No vascular surgery was undertaken at that time as the ulcer healed progressively.

Fungal infections develop as a result of poor foot hygiene, hyperhidrosis, and accumulation of moist detritus in the webs (Figure 8.6 shows another patient). *Interdigital tinea pedis* is the most common form of chronic fungal foot infection. Itching, redness, scaling, erosion and soaking of the skin with fluid usually occur, while in the late phase the redness subsides. *Trichophyton metagrophytes Trichophyton rubrum* or *Epidermophyton floccosum* may be found.

Topical terbinafine cream cures most infections caused by *Dermatophyta*, and should be continued for 2 weeks after symptoms subside.

Superficial bacterial infections in the interdigital spaces may cause thrombosis of the adjacent digital arteries and spread to deeper structures through the lumbrical tendons. Furthermore, edema impedes foot circulation, especially in the presence of peripheral vascular disease. Adequate foot hygiene and treatment of the fungal infection could prevent this complication.

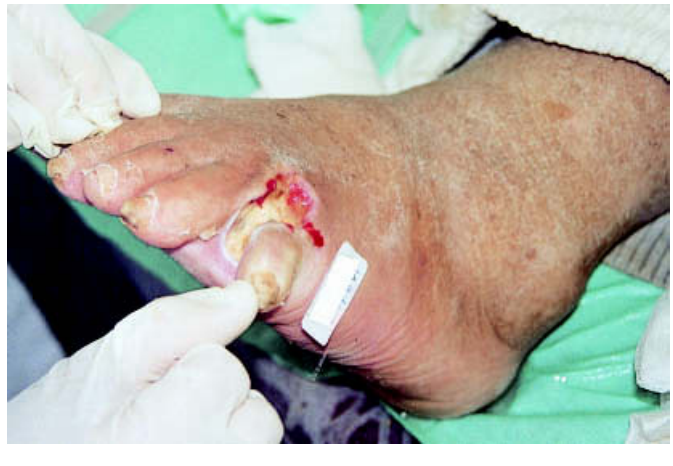

Figure 8.5 Full-thickness, infected neuro-ischemic ulcer in the fourth web space with exposure of the subcutaneous tissue. Ankle edema due to diabetic nephropathy, and onychodystrophy can be seen

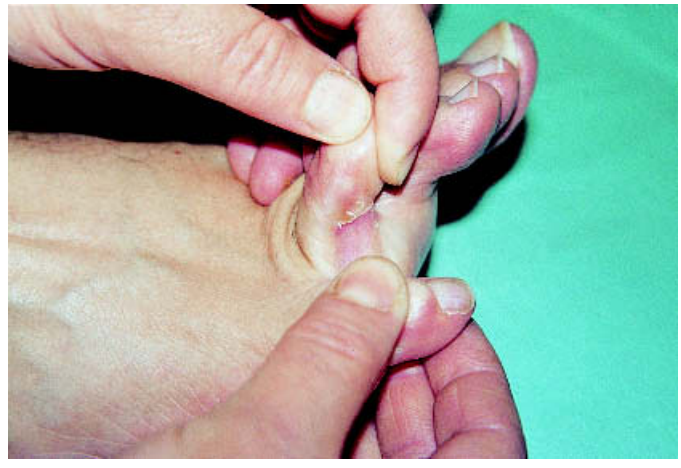

Figure 8.6 Fungal infection in the interdigital space with claw toe deformity

Keywords: Web space; infection; fungus infection; chronic tinea pedis; *Trichophyton metagrophytes Trichophyton rubrum* or *Epidermophyton floccosum*.

ONYCHOMYCOSIS

The patient whose feet are shown in Figures 5.12–5.16 was suffering from onychomycosis and the nails and nail beds were completely destroyed by fungus as shown in Figure 8.7. Onychomycosis *per se* does not cause foot problems, but when it affects the proximal nail (proximal subungual onychomycosis) it may cause chronic paronychia and serve as a portal for bacteria, resulting in deep tissue infection. It often co-exists with mycosis of the web spaces and it may be superinfected by bacteria, leading to deep tissue infection as well.

This patient was treated with terbinafine hydrochloride, both systemic (tablets 250 mg once daily) and topical (cream), for 3 months and was instructed in the appropriate foot care.

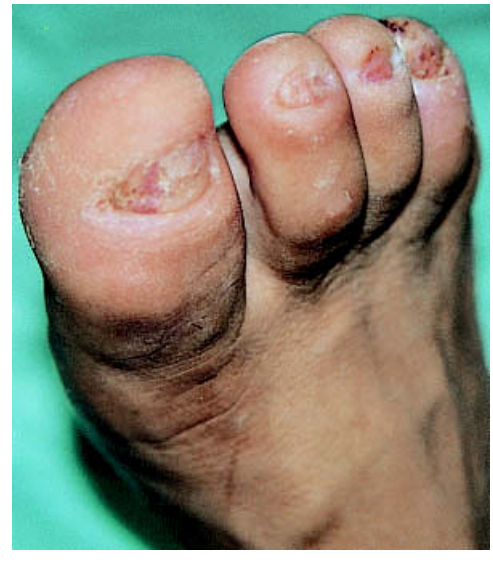

Figure 8.7 Fungal infection affecting all nails in addition to claw toes. Patient whose feet are shown in Figures 5.12–5.16

Chronic onychomycosis is classified into three clinical types: *distal subungual onychomycosis* is the most common form. The distal edge of the nail becomes infected and a yellow discoloration, onycholysis

and subungual debris develop. In p*roximal subungual fungal infection*, the second commonest form, *Trichophyton rubrum* accumulates hyperkeratotic debris under the nail plate and loosens the nail, eventually separating it from its bed. This fungus infects the underlying matrix and nail plate leaving the nail surface intact. *Leuconychia mycotica*, caused by *Trichophyton metagrophytes*, infects the nail superficially. The nail surface becomes dry, soft and friable but the nail remains attached to its bed. In addition to these fungi *Epidermophyton floccosum* may also be isolated from infected areas.

Itraconazole and fluconazole are also effective in the treatment of chronic onychomycosis.

Keywords: Onychomycosis; distal subungual onychomycosis; proximal subungual fungal infection; leuconychia mycotica; *Trichophyton metagrophytes*, *Trichophyton rubrum* or *Epidermophyton floccosum*; terbinafine; itraconazole; fluconazole

FUNGAL INFECTION WITH MULTIMICROBIAL COLONIZATION

Superficial ulcers of 10 days' duration on the facing sides of the left first and second toe of a 70-year-old type 2 diabetic lady with diabetic neuropathy, before debridement are shown in Figures 8.8 and 8.9. Note soaking of the skin. An X-ray excluded osteomyelitis. Staphylococcus coagulase-negative, *Pseudomonas aeruginosa* and enterobacteriaceae were recovered after swab cultures in addition to *Candida albicans*. She was treated successfully with itraconazole for 5 weeks. The patient used a clear gauze in order to keep her toes apart, together with local hygiene procedures twice daily. Weekly debridement was carried out and no antimicrobial agent was needed.

Keywords: Fungal infection

Figure 8.8 Neuro-ischemic ulcers facing each other on the first and second toe with fungal infection and soaked skin in addition to claw toes. Foot shown from the plantar aspect

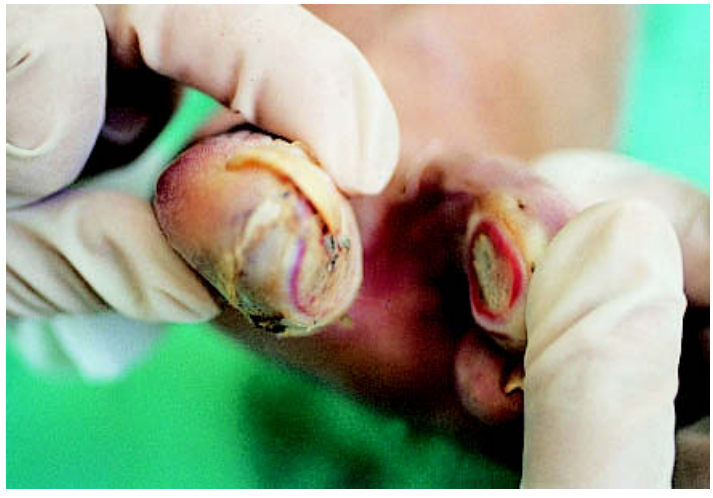

Figure 8.9 Neuro-ischemic ulcers facing each other on first and second toe, with fungal infection. Front aspect of Figure 8.8

DEEP TISSUE INFECTION AFTER INTERPHALANGEAL MYCOSIS

A 60-year-old female patient with type 2 diabetes diagnosed at the age of 47 years and treated with sulfonylurea and metformin and with poor glycemic control, was referred to the diabetic foot outpatient clinic because of a severe foot infection.

The patient had known mycosis between the fourth and the fifth toes of her right foot. Three days before her visit she noticed redness and mild pain on the dorsum of her toes. Her family doctor gave her cefaclor, but she became febrile and her foot became swollen, red and painful. No trauma was reported.

On examination, her foot was red, warm and edematous with pustules on its dorsum (Figure 8.10). The peripheral arteries were normal on palpation and peripheral neuropathy was present. Pathogen entry was probably via the area of the mycosis.

The patient was admitted to the hospital and treated with intravenous ciprofloxacin and clindamycin. No osteomyelitis was found on repeated radiographs. Extensive surgical debridement was carried out. Deep tissue cultures revealed *Staphylococcus aureus*, *Escherichia coli* and anaerobes. The patient was discharged in fair condition after a stay of 1 month.

Keywords: Mycosis; deep tissue infection

DEEP TISSUE INFECTION

A 50-year-old type 1 male diabetic patient with known diabetes since the age of 25 years was referred to the outpatient diabetic foot clinic for a large infected neuro-ischemic ulcer.

The patient suffered from retinopathy — treated with laser — established diabetic nephropathy, hypertension — treated with enalapril and furosemide — and severe neuropathy.

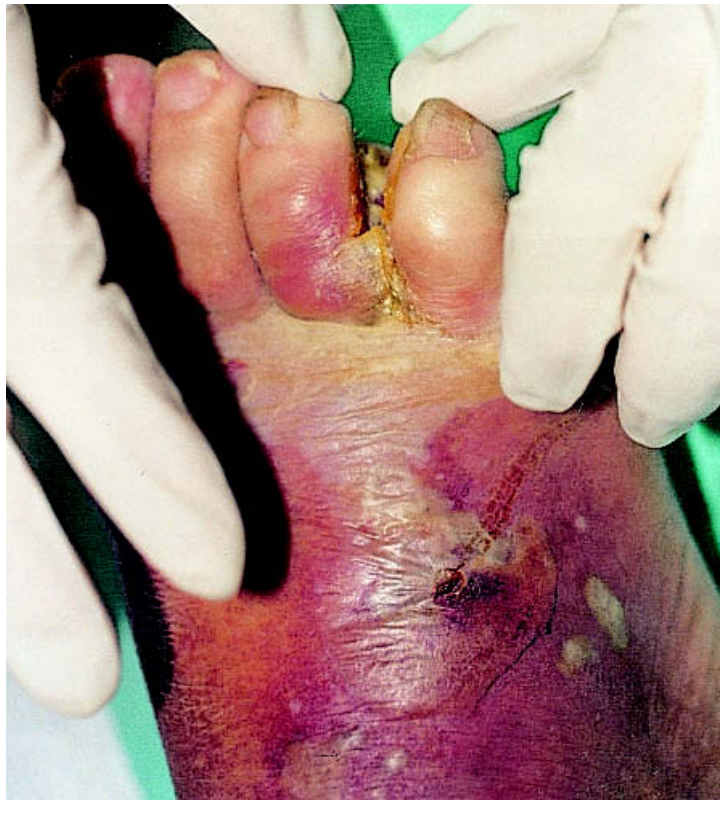

Figure 8.10 Deep tissue infection of the foot following web space mycosis. Redness and edema of the whole foot with pustules on the dorsum can be seen along with claw toes

Six months before visiting a surgeon, the patient had noticed a painless superficial ulcer caused by a new pair of shoes. Hoping it would subside quickly, he did not seek a doctor's advice and continued his daily activities although the ulcer became larger with surrounding erythema and eventually became purulent and odorous. Fever developed. A deep tissue culture revealed *Staphylococcus aureus, Klebsiella* spp. and anaerobes. Surgical debridement was carried out, and amoxicillin–clavulanic acid treatment was initiated. After 1 month of stabilization, with dressings being changed daily, the patient noticed increased purulent discharge and an intense foul odor.

On examination at the diabetic foot clinic, the patient was febrile and weak. He had complete loss of sensation. Peripheral pulses were palpable. Gross ankle and forefoot edema was noted and the short extensor of the toes and anterior tibial tendons was exposed (Figure 8.11). The common tendon sheath and subcutaneous tissue were necrosed. An acrid odor emanated from the foot even before the bandages were removed. A seropurulent discharge was being emitted from deeper structures. The patient was referred back to his surgeon; admission to the hospital and intravenous antibiotics together with extensive debridement followed, and due to abiding

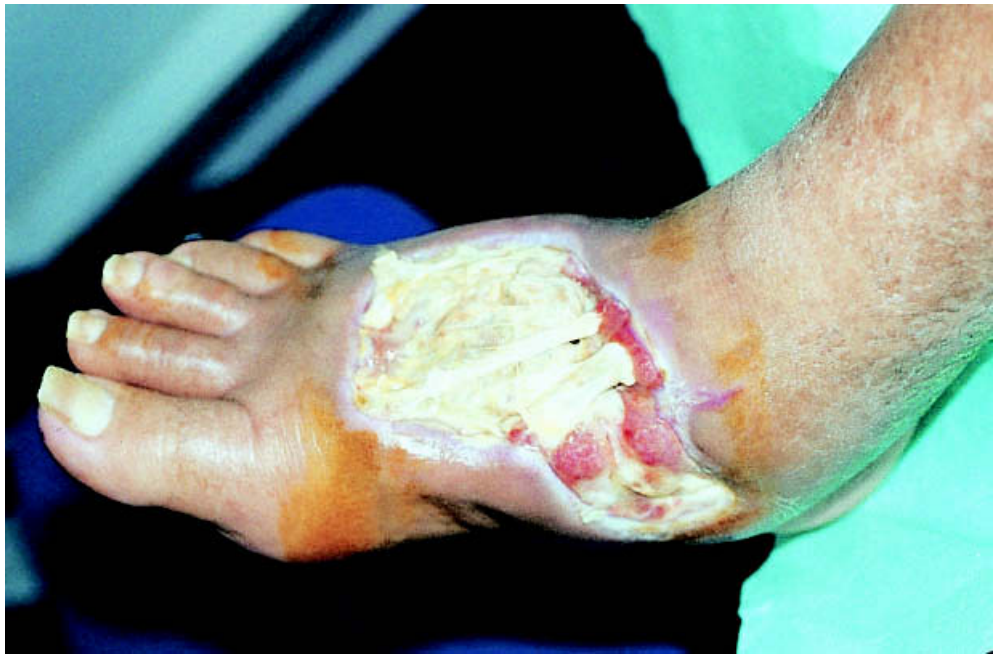

Figure 8.11 Deep tissue infection of the foot with gross ankle and forefoot edema. The short extensor of the toes and the anterior tibial tendons are exposed, while the common tendon sheath and subcutaneous tissue are necrosed

septic fever and the critical condition of the patient, a below-knee amputation was undertaken 2 days later.

Keywords: Deep tissue infection; amputation

DEEP TISSUE INFECTION OF A CHARCOT FOOT WITH A NEUROPATHIC ULCER

A 65-year-old female patient with type 2 diabetes mellitus since the age of 40 years attended the diabetic foot clinic because of a large ulcer of the sole of her left foot. She was being treated with insulin resulting in acceptable diabetes control (HbA_{1c}: 7.28%). She had a history of hypothyroidism as well as a history of ulcers under her right foot at the age of 63 years, which had healed completely.

The present ulcer had developed after a minor trauma to the sole of her foot while walking barefoot during the summer. It evolved within a month together with a fast progressing gross deformity of the foot. The patient complained of mild discomfort but no pain, so she kept on using both feet without any means of reducing the pressure on her ulcerated foot. She was treated with amoxicillin–clavulanic acid and clindamycin for 20 days.

On examination, her left foot was swollen, with midfoot collapse; it was warm (2.5 °C temperature difference to the contralateral foot), and crepitus was heard on passive movement. A large neuropathic

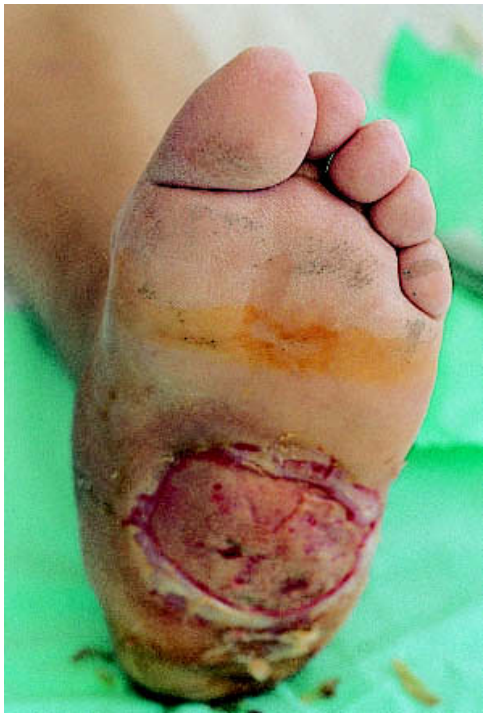

Figure 8.12 Neuro-osteoarthropathy. A large neuropathic non-infected ulcer surrounded by callus occupies the midsole

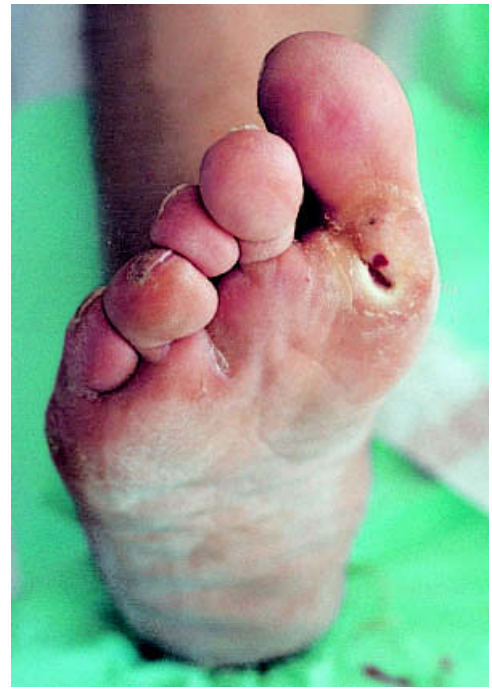

Figure 8.13 Right foot of the patient whose left foot is shown in Figure 8.12. A small, full-thickness neuropathic ulcer within an area of callus is present over the right first metatarsal head

non-infected ulcer of size $8 \times 7 \times 0.4$ cm occupied the midsole surrounded by callus (Figure 8.12). A small, full-thickness neuropathic ulcer was present within an area of callus formation over the right first metatarsal head (Figure 8.13). The skin on both her feet was dry and the peripheral pulses were palpable. The vibration perception threshold was 20 V in both feet. Monofilament sensation was absent, as were sensations of light touch, pain and temperature perception.

Debridement was carried out; an X-ray showed disruption of the tarsometatarsal joint (Lisfranc's joint), bone absorption of the first and second cuneiforms and dislocation of the cuboid bone (Figure 8.14). A diagnosis of acute neuro-osteoarthropathy was made and a single dose of 90 mg of pamidronate was administered. The presence of ulcers prevented the use of a total-contact cast since daily changes of dressings were needed. The patient was instructed to refrain from walking and to visit the diabetic foot clinic on a weekly basis. After 1 month the midsole ulcer was smaller compared to its initial size (Figure 8.15) and showed no signs of infection. The ulcer under her right sole healed. There was no difference in the temperature between the two feet.

After an absence of 3 weeks the patient visited the clinic with acute foot infection and fever. The midsole ulcer was

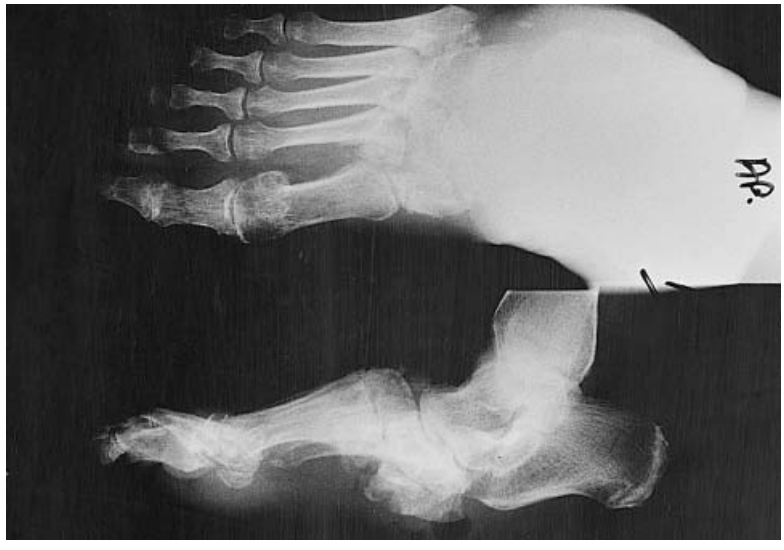

Figure 8.14 Plain radiographs showing neuro-osteoarthropathy in the left foot of the patient whose feet are illustrated in Figures 8.12 and 8.13. Disruption of the tarsometatarsal joint (Lisfranc's joint), resorption of the first and second cuneiforms and midfoot collapse can be seen

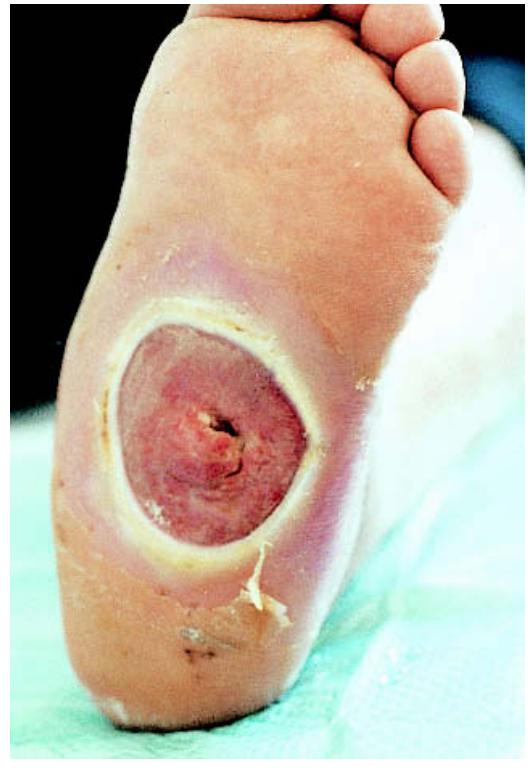

Figure 8.15 Left neuro-osteoarthropathic foot of the patient whose feet are shown in Figures 8.12–8.14. Progress of the plantar neuropathic ulcer after 1 month of chiropody treatment. Healthy granulated tissue covers the bed of the ulcer

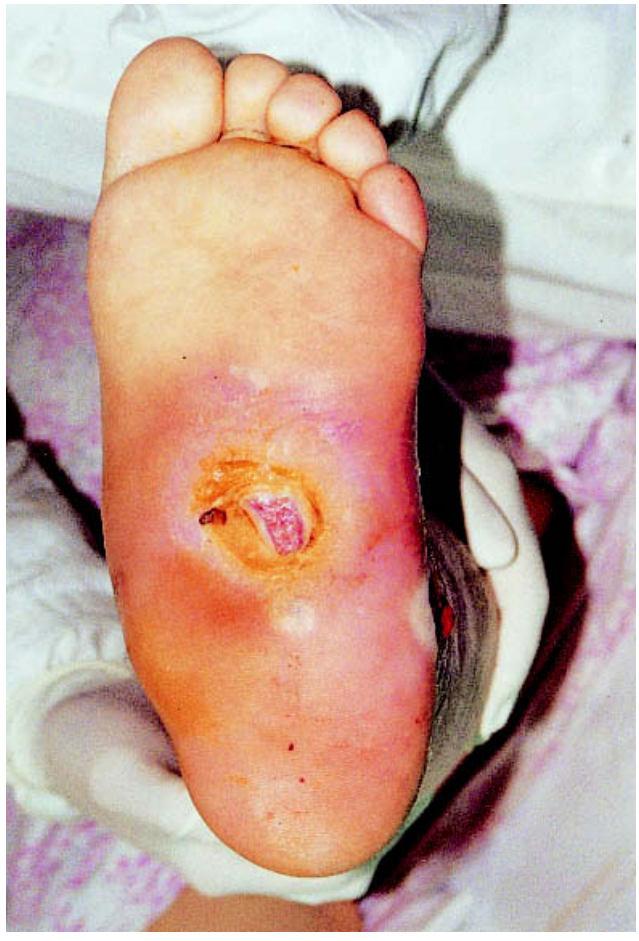

Figure 8.16 Left neuro-osteoarthropathic foot of the patient whose feet are shown in Figures 8.12–8.15, 3 weeks after the photograph shown in Figure 8.15 was taken. Signs of infection (cellulitis, blisters and edema) are present

much smaller (Figure 8.16), surrounded by cellulitis, and a new infected ulcer was present on the lateral aspect of the hindfoot (Figure 8.17). The patient insisted that she had complied with the instructions, except for the last week, when she felt confident that the ulcer had healed. She was admitted to the hospital and underwent extensive surgical debridement. Intravenous antibiotics (ciprofloxacin, penicillin and clindamycin) were administered but the high fever persisted despite treatment; the infection spread to the lower tibia and the patient became septic. On the 10th day of hospitalization, the critical condition of the patient necessitated a below-knee amputation. She was discharged in good clinical condition after 1 week.

Keywords: Deep tissue infection; acute neuro-osteoarthropathy; neuropathic ulcer; below-knee amputation

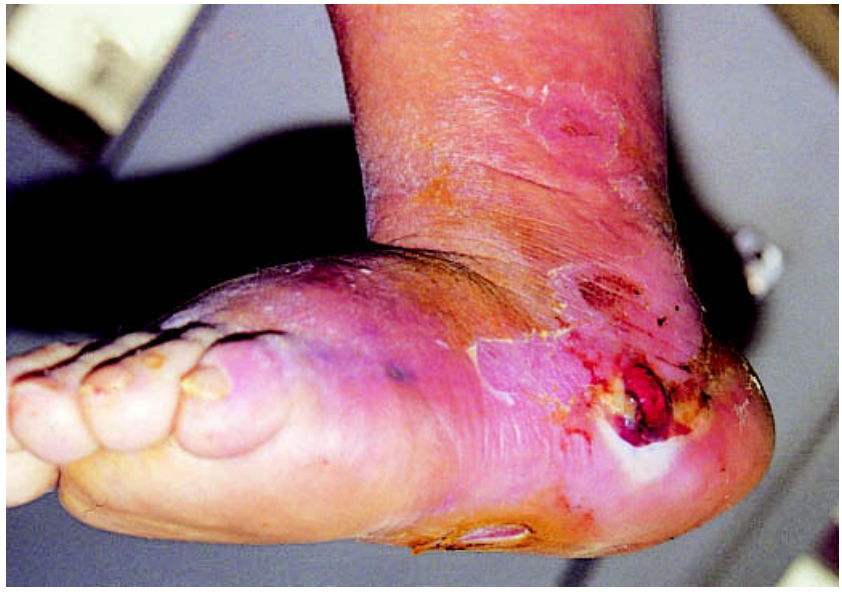

Figure 8.17 Lateral aspect of the foot shown in Figure 8.16. Infection has spread to the whole foot and the lower tibia. The superficial ulcer on the lateral aspect of the hindfoot may have been caused by rupture of a blister

OSTEOMYELITIS

A 69-year-old female patient with type 2 diabetes diagnosed at the age of 54 years and treated with sulfonylurea, was referred to the outpatient diabetic foot clinic for an infection of her right second toe. She had background diabetic retinopathy and hypertension. She complained of numbness and a sensation of pins and needles in her feet at night.

On examination, she had findings of severe neuropathy (no feeling of light touch, pain, temperature, vibration or a 5.08 monofilament; Achilles tendon reflexes were absent; the vibration perception threshold was >50 V in both feet). Peripheral pulses were weak and the ankle brachial index was 0.7. Dry skin and nail dystrophies were present. A superficial ulcer with a sloughy base was seen on the dorsum of her right second toe which was red, swollen and painful, having a sausage-like appearance (Figure 8.18). She did not mention any trauma, but inspection of her shoes revealed a prominent seam inside the toe box of her right shoe.

The sausage-like appearance of a toe usually denotes osteomyelitis. Bone infection was confirmed on X-ray, showing osteolysis of the first and second phalanges. *Staphylococcus aureus* and *Klebsiella pneumoniae* were cultured from the base of the ulcer. The patient was treated with cotrimoxazole and clindamycin for 2 months. She was also referred to the Vascular Surgery Department for a percutaneous transluminal angioplasty of her right popliteal artery. After 2 months the ulcer was still active and the patient had local extension of osteomyelitis despite the restoration of the circulation in the periphery. She eventually had her second

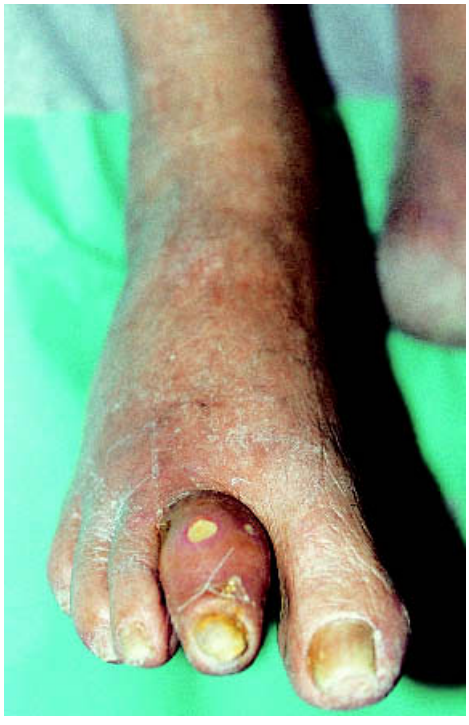

Figure 8.18 Sausage-like toe deformity usually denotes underlying osteomyelitis

ray amputated. A bone culture revealed the presence of *Staphylococcus aureus*. She continued with cotrimoxazole for two more weeks.

Keywords: Osteomyelitis; painful–painless feet

OSTEOMYELITIS OF THE HALLUX

A 30-year-old male patient with type 1 diabetes diagnosed at the age of 11 years was admitted because of infected foot ulcers on his right hallux. He had a mild fever and a history of proliferative diabetic retinopathy and microalbuminuria. Diabetes control was poor (HBA_{1c}: 9.5%). He reported a trauma to his left foot 2 months earlier when an object fell on his feet while working. A superficial ulcer had developed on the dorsal aspect of his right great toe; the ulcer had become infected because the patient felt no pain and therefore did not seek medical advice.

On examination, pedal pulses were normal. Severe peripheral neuropathy was found and the vibration perception threshold was 30 V in both feet. An infected right hallux with purulent discharge, necrotic tissue at the tip, and cellulitis were observed (Figure 8.19). A plain radiograph showed osteomyelitis involving both distal phalanges (Figure 8.20).

A culture of the pus revealed *Pseudomans maltophila, Enterobacter cloacae* and

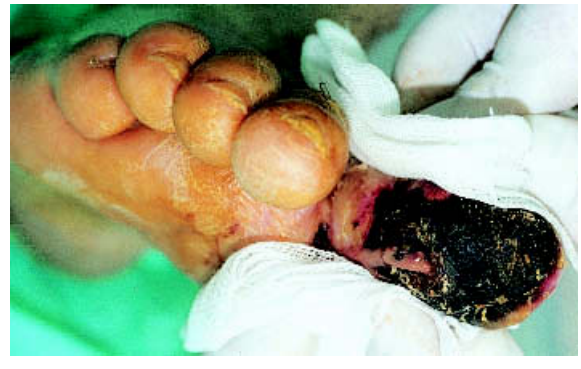

Figure 8.19 Infection of the hallux with purulent discharge, necrotic tissue at the tip and cellulitis

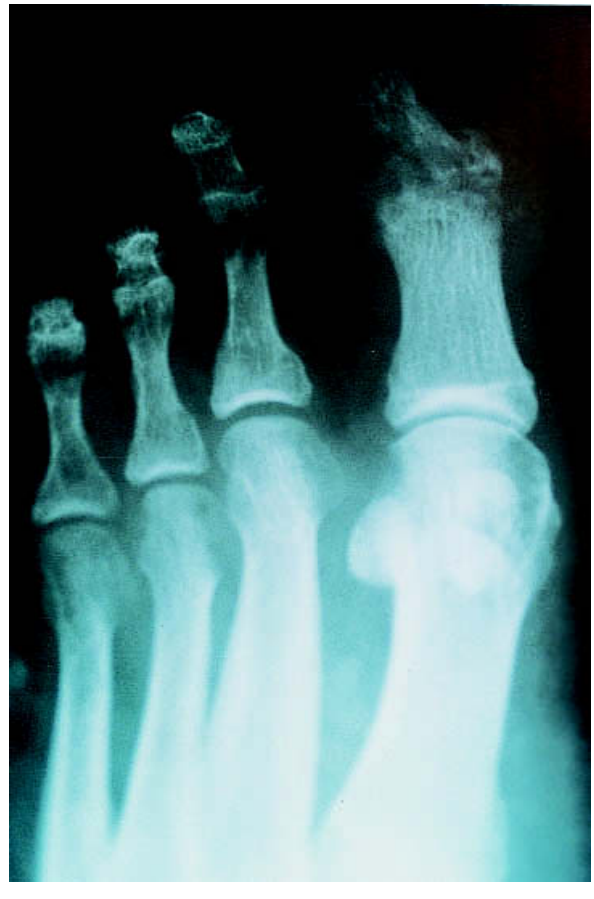

Figure 8.20 Osteolysis of the distal phalanx and condyle of the proximal phalanx due to osteomyelitis of the hallux. Plain radiograph of the foot shown in Figure 8.19

anaerobes, and the patient was treated with ciprofloxacin and ampicillin–sulbactam for 2 weeks, based on the antibiogram. An amputation of the right great toe was undertaken due to persistent osteomyelitis.

Keywords: Hallux; osteomyelitis; amputation

PHLEGMON

A 62-year-old male diabetic patient with type 2 diabetes diagnosed at the age of 42 years and treated with sulfonylurea, biguanide and acarbose and whose diabetes control was acceptable, visited the outpatient diabetic foot clinic due to infection of the sole of his right foot. He had hypertension and coronary heart disease treated with metoprolol and aspirin. He had no previous history of foot problems.

On examination, the patient had fever, severe diabetic neuropathy, and bounding pedal pulses. He had hallux valgus, claw toes, prominent metatarsal heads, onychodystrophy and dry skin. Callus formation superimposed on a neuropathic ulcer over his third metatarsal head was present; a callus was also noted over his fifth metatarsal head. A superficial, painless,

infected ulcer with purulent discharge was present under Lisfranc's joint (Figure 8.21). This infection progressed to a phlegmon 2 days after a minor shear trauma.

The patient was admitted, and intravenous amoxicillin–clavulanate was initiated. A plain radiograph excluded osteomyelitis or gas collection within the soft tissues. Computerized tomography revealed a phlegmonous subcutaneous mass under the base of the metatarsals (Figure 8.22). A sterile probe was used to detect any sinuses or abscesses, but none was found. The patient remained bedridden for 1 week and the infection subsided. He continued antibiotics for one more week with limited mobilization and he was released from hospital in excellent condition. Oral antibiotics were continued for two more weeks. Preventive footwear was prescribed and the

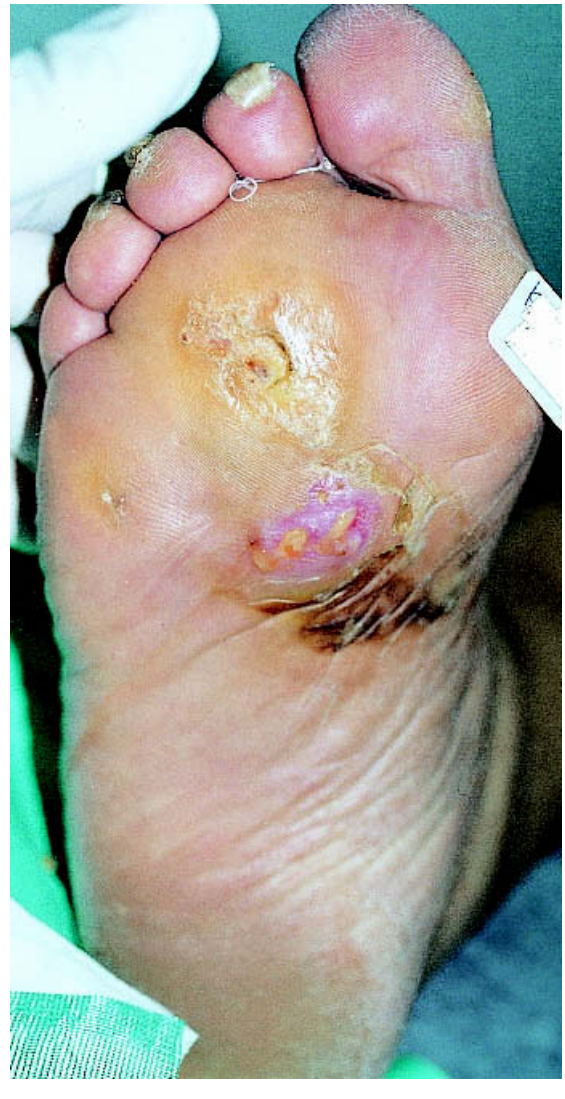

Figure 8.21 Superficial infected ulcer with purulent discharge under Lisfranc's joint. Callus formation is superimposed on neuropathic ulcer over the third metatarsal head with callus formation over the fifth metatarsal head. Hallux valgus, claw toes, prominent metatarsal heads, onychodystrophy and dry skin can be seen

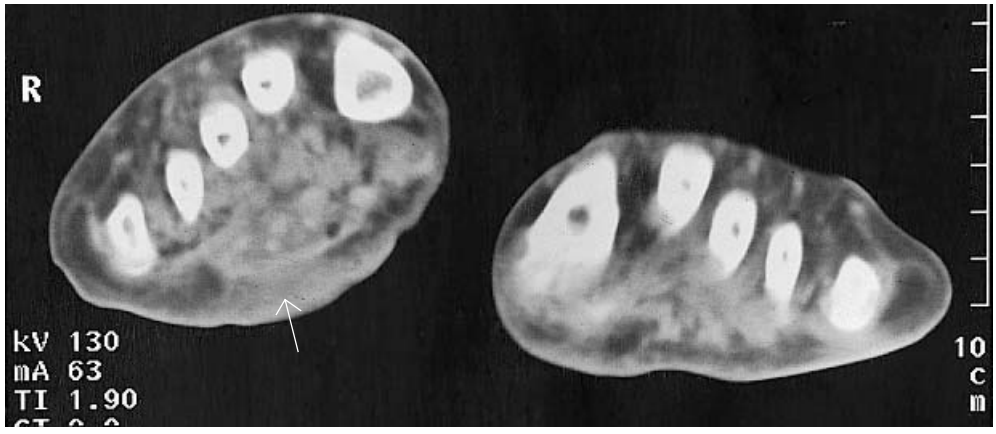

Figure 8.22 Computerized tomography of the feet of the patient whose right foot is shown in Figure 8.21. A phlegmonous subcutaneous mass is present under the base of the metatarsals (arrow)

patient continued to visit the outpatient diabetic foot clinic on a regular basis.

Computerized tomography is useful in identifying areas of phlegmon within the soft tissues. It may provide information about the exact anatomic location and extent, so that aspiration or surgical drainage can be undertaken. Magnetic resonance imaging and ultrasound studies are also helpful in this respect.

Keywords: Neuropathic ulcer; computerized tomography; phlegmon

INFECTED PLANTAR ULCER WITH OSTEOMYELITIS

A 50-year-old female diabetic patient with type 2 diabetes diagnosed at the age of 44 years and treated with sulfonylurea, was referred to the outpatient diabetic foot clinic because of a chronic infected ulcer on her left foot. The patient lived alone and she was being treated for depression; she had good diabetes control. A minor trauma under her left foot was reported to have occurred 2 years previously. She had treated the injury with different types of gauzes and creams, but it failed to heal. She presented to the clinic with a large, painless, infected ulcer under her left foot (Figure 8.23).

On examination, an irregular, soaked, foul-smelling ulcer with sloughy bed, and surrounding cellulitis of 3 cm in diameter was found; body temperature was normal. Diabetic neuropathy was diagnosed, while peripheral pulses were normal. Signs of osteomyelitis (osteolysis of the first metatarsal head, and the base of proximal phalanx of the hallux, with periosteal reaction) were noted on the radiograph (Figure 8.24). A post-debridement swab culture from the base of the ulcer revealed methicillin-resistant *Staphylococcus aureus* and *Escherichia coli*. The patient was admitted to the hospital. The white blood cell count was 14,700/mm^3, anemia (Hb: 9.8 g/dl) characteristic of chronic disease was found, the erythrocyte sedimentation rate was 90 mm/h and the level of C-reactive protein was 70 mg/dl. She was treated with 600 mg teicoplanin

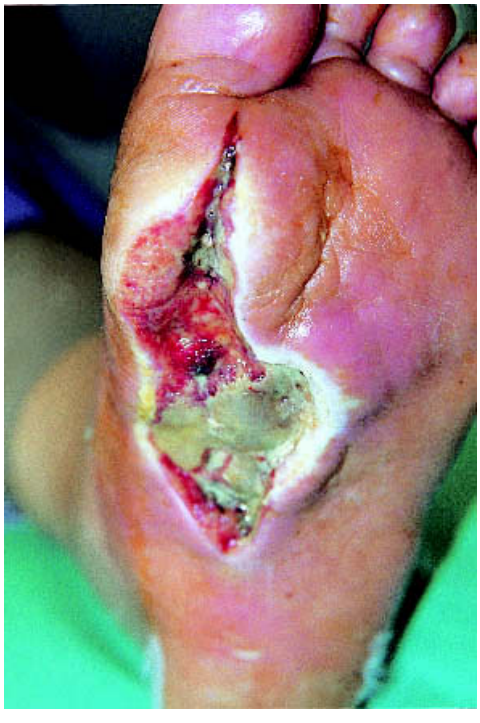

Figure 8.23 A large, irregular, soaked and infected neuropathic ulcer with sloughy bed and surrounding cellulitis of 3 cm in diameter is shown here. A minor trauma reported to have occurred 2 years earlier was the cause of this ulcer

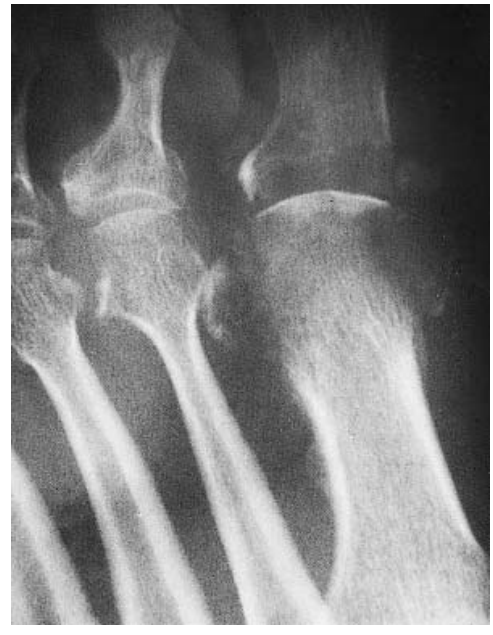

Figure 8.24 Osteolysis of the first metatarsal head and the base of proximal phalanx of the hallux with periosteal reaction due to osteomyelitis are shown on this plain radiograph of the foot illustrated in Figure 8.23

intravenously once daily and the ulcer was debrided and dressed. The cellulitis progressively subsided, the ulcer became clear and healthy granulating tissue began to cover the ulcerated area (Figure 8.25). The patient was discharged from the hospital in good clinical condition. She continued treatment with intramuscular teicoplanin for three more months and attended the outpatient diabetic foot clinic on a weekly basis. Complete offloading of pressure from the ulcerated area was achieved by the use of a wheelchair for most of her activities. Platelet-derived growth factor-β (becaplermin) was applied once daily. The ulcer diminished progressively (Figure 8.26) and healed in 4 months; no relapse occurred.

All patients with deep or long-standing ulcers should be evaluated for osteomyelitis. The possibility of an ulcer being complicated by osteomyelitis increases when the diameter of the ulcer exceeds 2 cm and the depth is greater than 3 mm; the possibility of complications becomes even higher when the white blood cell count, the erythrocyte sedimentation rate and the C-reactive protein levels are high.

Treatment of acute osteomyelitis includes parenteral administration of antibiotics for 2 weeks initially, and the continuation of oral treatment for a prolonged period (at least 6 weeks).

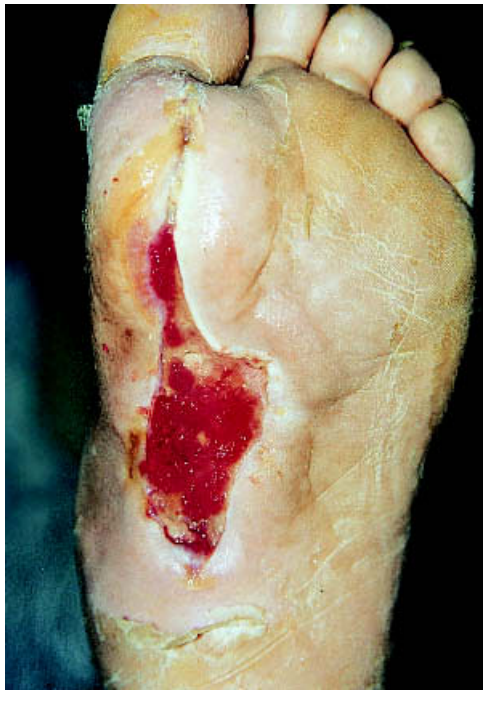

Figure 8.25 Clear ulcer with healthy granulating tissue after 1 month of appropriate treatment in the patient whose foot is shown in Figures 8.23 and 8.24

Keywords: Neuropathic ulcer; acute osteomyelitis; platelet-derived growth factor-β (PDGF-β, becaplermin)

NEUROPATHIC ULCER WITH OSTEOMYELITIS

A 57-year-old obese male patient with type 2 diabetes diagnosed at the age of 40 years was referred to the outpatient diabetic foot clinic because of a chronic ulcer under his right foot. He was being treated with insulin and metformin with acceptable diabetes control (HBA_{1c}: 7.8%). He had a history of background retinopathy and cataract in both eyes. He reported a severe deep tissue infection 5 years earlier after a burn sustained under his right foot. At that time he was hospitalized for about 1 month and treated with intravenous antibiotics and surgical debridement.

On examination, the patient had severe diabetic neuropathy with loss of sensation of pain, light touch, temperature, vibration,

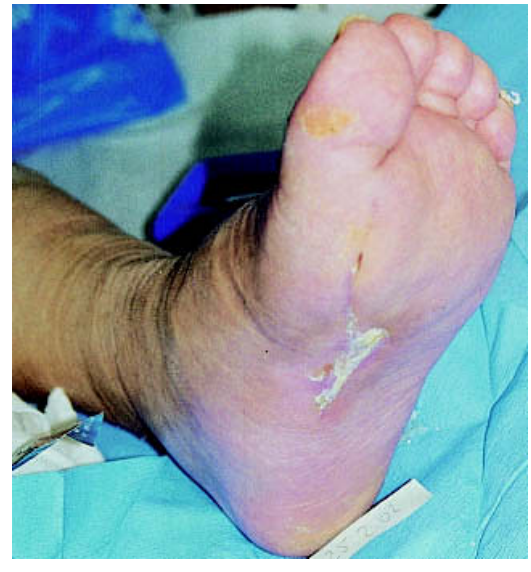

Figure 8.26 Healing of the ulcer affecting the foot shown in Figures 8.23–8.25. This photograph was taken 3 months after that shown in Figure 8.25

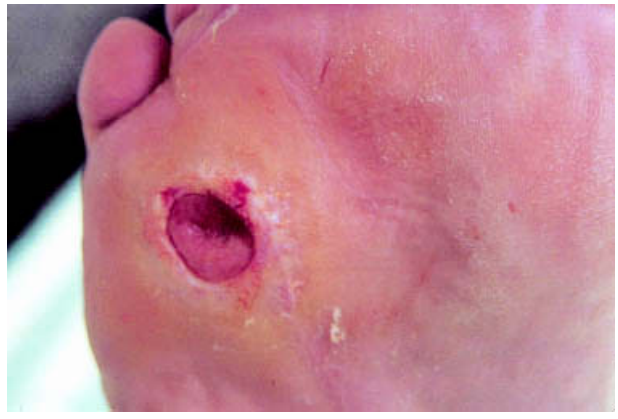

Figure 8.27 Full-thickness neuropathic ulcer post-debridement under a prominent fourth metatarsal head

Figure 8.28 Commercially-available extra depth therapeutic shoe

and 5.07 monofilaments. Achilles tendon reflexes were absent. The vibration perception threshold was above 50 V bilaterally, while the peripheral pulses were normal. A scar was noted on the dorsum of his right foot which had an overriding fourth toe, as a result of past surgical procedures. A full-thickness neuropathic ulcer was present under his fourth metatarsal head surrounded by callus (Figure 8.27). A bony prominence could also be felt under the ulcerated area. A plain radiograph did not show osteomyelitis or neuro-osteoarthropathy. Debridement of the ulcer was carried out and extra depth therapeutic shoes with a flat insole were prescribed (Figure 8.28); a window was made in the insole in order to offload pressure on the ulcerated area; the ulcer began to heal well (Figure 8.29).

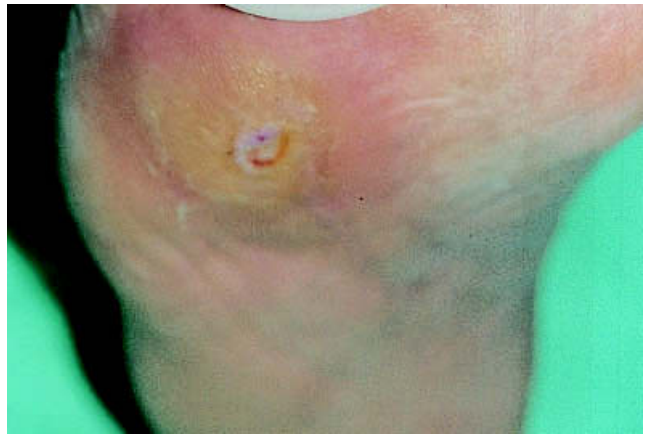

Figure 8.29 Healing of the neuropathic ulcer shown in Figure 8.27 pre-debridement

The patient kept himself very active. He returned to the clinic after 3 weeks absence with a deeper ulcer involving the tendons (Figure 8.30). The underlying bone could not be detected with a sterile metal probe and a plain radiograph did not show osteomyelitis. An elevated erythrocyte sedimentation rate (74 mm/h) and mild leukocytosis were found, therefore the possibility of osteomyelitis was high. A magnetic resonance imaging-T1-weighted sagittal image of the foot was obtained, showing a phlegmonous mass starting from the skin and extending to the deeper tissues causing erosion of the fourth metatarsal head (Figure 8.31). The patient was hospitalized so that offloading pressure from the ulcerated area was enforced, and intravenous antibiotics were administered. Two weeks later the size of the ulcer had decreased by almost 50%.

Several methods are used for the diagnosis of osteomyelitis. *Probe-to-bone tests* (contacting the bone with a sterile metal probe) have a sensitivity of more than 90% and they are carried out at the bedside. *Plain radiographs* have a sensitivity of 55%, but when repeated — usually 2 weeks later — the sensitivity is higher, making this the most cost-effective diagnostic procedure. *Computerized tomography* may reveal areas with subtle abnormalities such as periosteal reactions, small cortex erosions and soft tissue abnormalities. *Magnetic resonance imaging* has a sensitivity of almost 100% and a specificity of over 80% and has the potential to reveal abscesses. Therefore this is the preferred method for the diagnosis of osteomyelitis in many centers in cases where the plain radiographs do not provide sufficient information to make a conclusive diagnosis. However, the specificity of MRI decreases in the presence of neuro-osteoarthropathy, prior bone biopsy, recent bone fracture or recent surgery. *Magnification radiography* is also a very useful method for the detection of early osteomyelitis and it is used to follow up the disease.

Bone scintigraphy imaging is explained in Figure 8.37.

Keywords: Neuropathic ulcer; magnetic resonance imaging; MRI; osteomyelitis; diagnostic methods for osteomyelitis

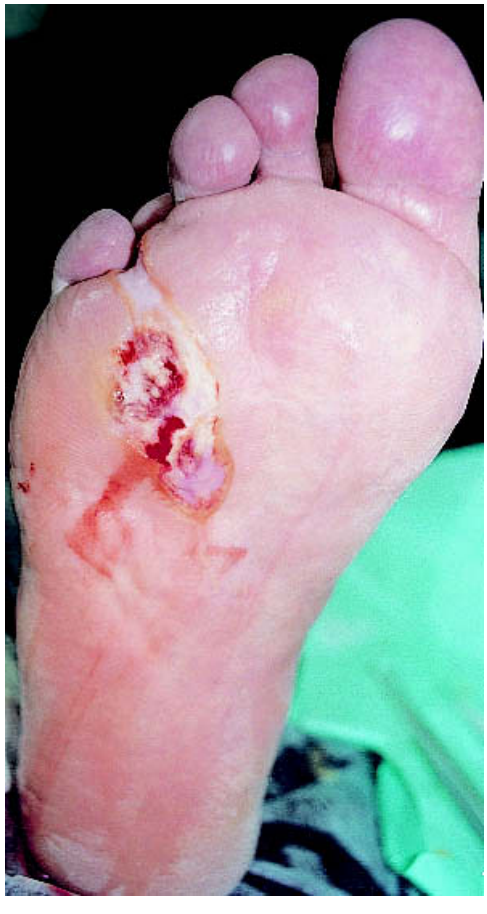

Figure 8.30 The neuropathic ulcer shown in Figures 8.27 and 8.29 has been aggravated by the patient's refusal to reduce activity levels and poor compliance with measures to offload pressure from the affected area

OSTEOMYELITIS OF THE FIRST METATARSAL HEAD

A 74-year-old male patient with type 2 diabetes attended the outpatient diabetic foot clinic because of a chronic painless ulcer on the medial aspect of the right first metatarsal head (Figure 8.32). The ulcer developed over a bunion deformity, and had persisted for 10 months.

On examination, the peripheral pulses were palpable and the patient had severe peripheral neuropathy. He could not feel pain, light touch, vibration or 5.07 monofilaments. The vibration perception threshold was above 50 V in both feet. After debridement, the underlying bone could be felt by means of a sterile probe. A plain radiograph revealed osteomyelitis of the first metatarsal head and the proximal phalanx of the right great toe (Figure 8.33). The patient sustained a first ray amputation.

Chronic osteomyelitis needs surgical removal of the infected bone. However, recent data suggest that prolonged treatment with antibiotics (for 1 or 2 years) may eradicate chronic osteomyelitis. However, no consensus on this issue exists at present.

Keywords: Chronic osteomyelitis; first ray amputation; neuropathic ulcer

CHRONIC NEUROPATHIC ULCER WITH OSTEOMYELITIS

A 46-year-old male patient with type 1 diabetes diagnosed at the age of 27 years was referred to the outpatient diabetes foot clinic because of a chronic ulcer under his right fifth metatarsal head. He had acceptable diabetes control (HBA_{1c}: 7.7%), proliferative diabetic retinopathy treated with laser in both eyes, but no nephropathy. He complained of muscle cramps during the night and chronic constipation interrupted by episodes of nocturnal diarrhea. The patient had a history of painless diabetic foot ulceration for 3 years under his right foot after a burn injury. He had attended the surgery department of a country hospital,

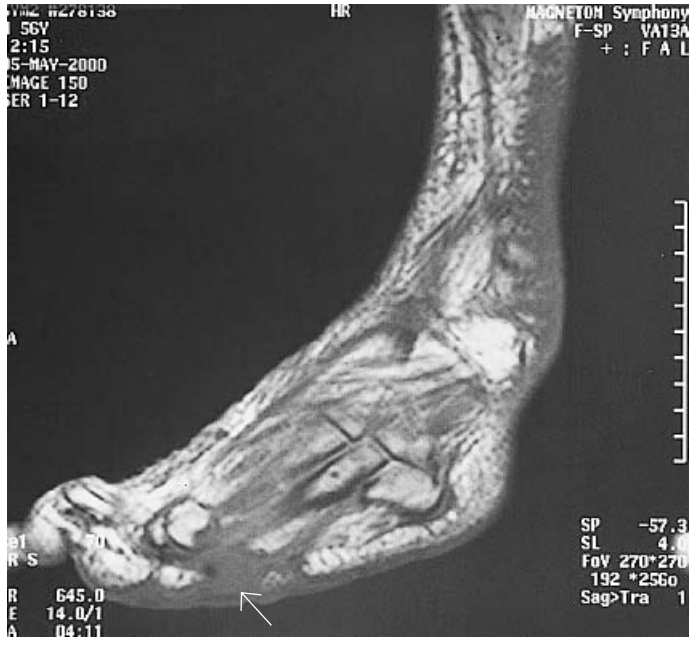

Figure 8.31 MRI image showing osteomyelitis. A magnetic resonance imaging-T1-weighted sagittal image of the foot illustrated in Figure 8.30 showing a phlegmonous mass (arrow) extending from the skin into the deeper tissues and causing erosion of the fourth metatarsal head

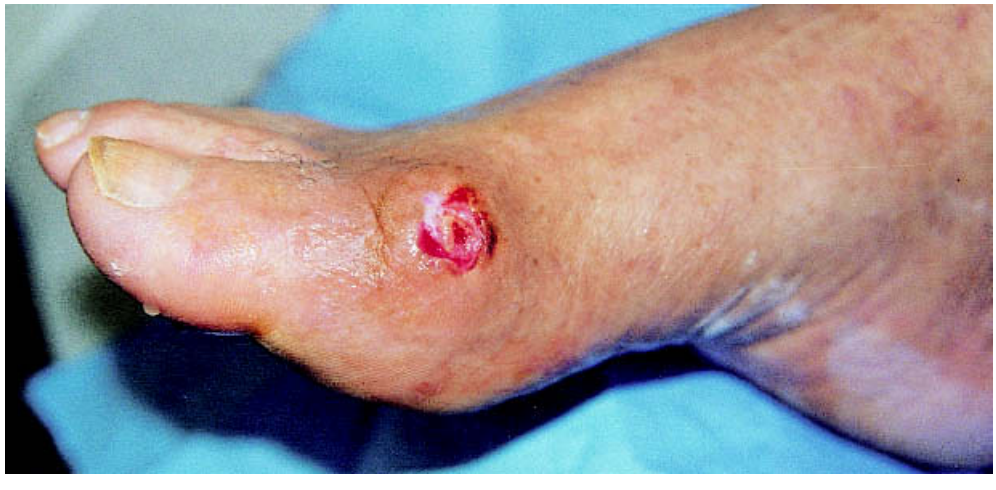

Figure 8.32 Chronic neuropathic ulcer over a bunion deformity

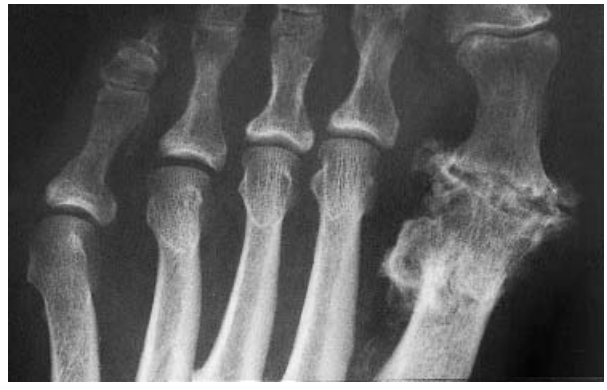

Figure 8.33 Plain radiograph of the foot illustrated in Figure 8.32 showing bone resorption, periosteal reaction and destruction of metatarsophalangeal joint of the hallux due to osteomyelitis

where he had his foot dressed and several courses of antibiotics were prescribed. The patient continued to keep himself active, without any special footwear since he felt no discomfort or pain.

On examination, severe diabetic neuropathy was found. The peripheral pulses were palpable and a full-thickness neuropathic ulcer with gross callus formation was observed under his right fifth metatarsal head (Figure 8.34). Sharp debridement was carried out and the underlying bone was probed with a sterile probe. A plain radiograph revealed pseudoarthrosis of a stress fracture of the upper third of his fifth metatarsal, bone resorption in the metatarsophalangeal joint, and osteolytic lesions in the fifth metatarsal epiphysis (Figures 8.35 and 8.36). Post-debridement cultures from the base of the ulcer revealed *Staphylococcus aureus*, *Proteus vulgaris* and *Enterococcus* spp. The patient was treated with amoxicillin–clavulanic acid 625 mg three times daily for 2 weeks. He was advised to rest and appropriate footwear and insoles were prescribed. A fifth ray amputation was undertaken and antibiotics continued for two more weeks. A bone culture revealed *Staphylococcus aureus*. The wound healed completely in 2 weeks.

A ray amputation consists of removal of a toe together with its metatarsal. The uninvolved half of the fifth metatarsal shaft was preserved, so that it retained the insertion of the short peroneal muscle. Ray amputation results in narrowing of the forefoot, but the cosmetic and functional result is excellent. However, the biomechanics of the foot are disturbed after such an operation and this leads to the exertion of high pressure under the metatarsal heads of the adjacent rays.

Keywords: Neuropathic ulcer; osteomyelitis; ray amputation

BONE SCINTIGRAPHY IMAGING

The scintigraphy findings of a patient with possible osteomyelitis are discussed below and the history of this patient is illustrated in Figures 9.3 to 9.5 in Chapter 9.

A plain radiograph showed findings compatible with osteomyelitis or neuro-osteoarthropathy of the second and third

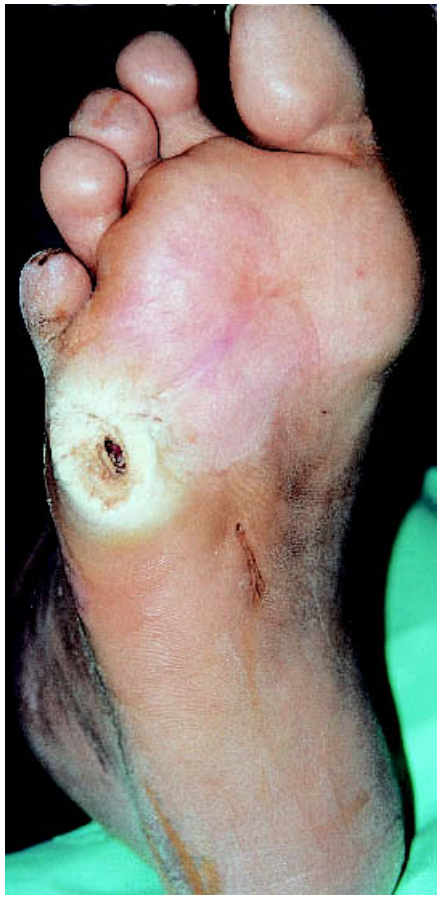

Figure 8.34 Full-thickness chronic neuropathic ulcer with gross callus formation under the right fifth metatarsal head

metatarsal heads of this female patient. She was referred for a technetium-99m (^{99}Tc) phosphonate scan. Images obtained during the *flow phase* are shown in the left upper panel of Figure 8.37; during this phase a series of 3-s image acquisitions of the site in question is obtained. They showed increased radionuclide uptake by the tarsometatarsal area of her left foot. A static blood pool image (*blood pool phase*) obtained 5 min later is shown in the right upper panel. A static delayed image (*delayed phase*) obtained 3 h later is shown in the left lower panel. All images showed increased uptake in the same areas. A gallium-67 citrate study performed on the same day (right lower panel of Figure 8.37), showed increased radionuclide uptake at the tarsometatarsal area. Based on the results of bone scintigraphy the patient was diagnosed as having osteomyelitis in the tarsometatarsal area.

^{99}Tc scintigraphy is useful in cases of questionable osteomyelitis. It has a high sensitivity (over 90%) but a low specificity (33%), particularly in the presence of neuro-osteoarthropathy. Although increased radionuclide uptake during the flow and pool phase is not specific to the diagnosis of osteomyelitis (it may mean soft tissue, bone infection or both), delayed images of the ^{99}Tc scintigraphy showed increased blood flow to the bones only, thus increasing the specificity of the method in the diagnosis of bone infection. Patients with neuro-osteoarthropathy have increased bone blood flow in the absence of osteomyelitis.

Like ^{99}Tc scintigraphy, gallium-67 citrate accumulates in both osteomyelitis and neuro-osteoarthropathy. This is the reason for its low specificity in the diagnosis of osteomyelitis in diabetic patients. Indium-111 white blood cell imaging (^{111}In WBCs) is expensive, time consuming, has poor spatial resolution and does not distinguish soft tissue from bone infection.

Keywords: Scintigraphy; bone scans; diagnosis of osteomyelitis

OSTEOMYELITIS OF THE HEEL

A 71-year-old female patient with type 2 diabetes was admitted to the hospital

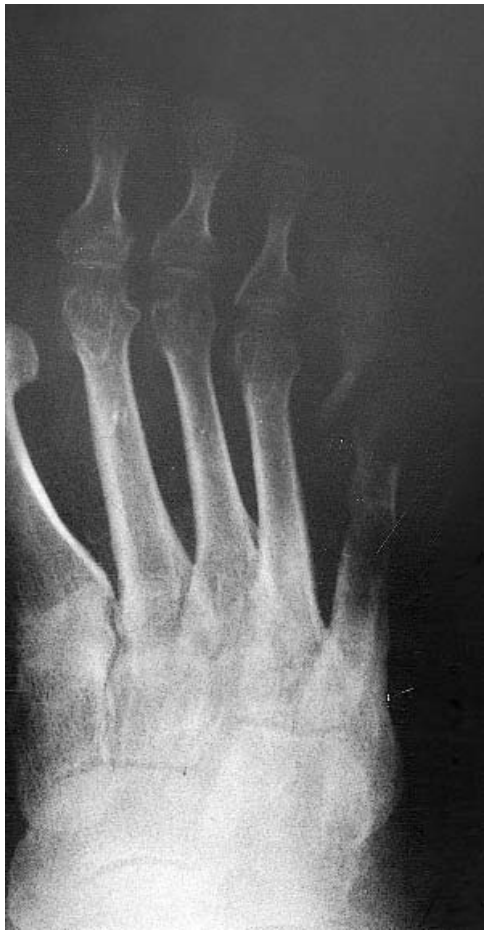

Figure 8.35 Anteroposterior plain radiograph of patient of Figure 8.34. Osteomyelitis. Pseudoarthrosis of a stress fracture of the upper third of the fifth metatarsal, bone resorption at the metatarsophalangeal joint, and osteolytic lesions at the fifth metatarsal epiphysis

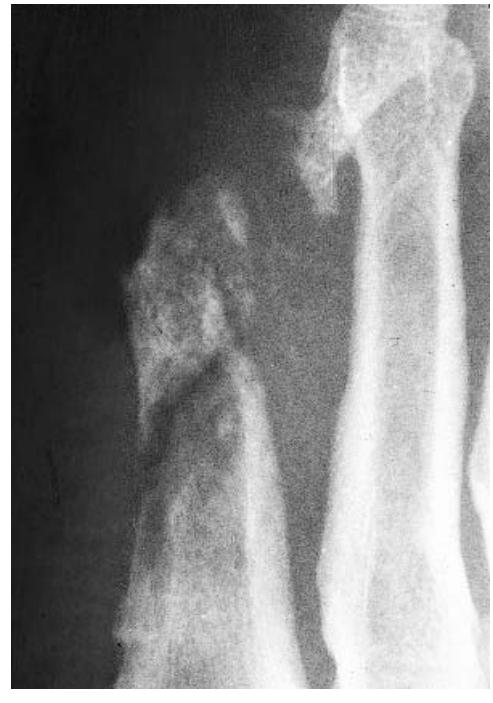

Figure 8.36 Lateral plain radiograph of foot shown in Figures 8.34 and 8.35, focused on the osteomyelitic lesion in the fifth metatarsal. Note that the phalanx of fifth toe continues over fourth metatarsal head

because of a severe infection of her right foot. She had a history of type 2 diabetes diagnosed at the age of 51 years, diabetic nephropathy, background diabetic retinopathy — treated with laser — hypertension and ischemic heart disease. She also had a history of stroke at the age of 69 years. A heel ulcer caused after the rupture of a blister under her right heel, which developed after walking in tight new shoes, had persisted for about 1 year. The ulcer progressively became deeper and larger. The patient reported two septic episodes with infection at the same site, for which she was hospitalized for prolonged periods.

On examination, her body temperature was 39.2 °C, blood pressure 90/50 mmHg, heart rate 120 beats/min and weak, and she was anuric. Her right foot and the tibia were warm, red and swollen. A large, foul-smelling, neuro-ischemic ulcer with gross purulent discharge was seen on the posterior surface of her right heel (Figure 8.38). The calcaneus was exposed.

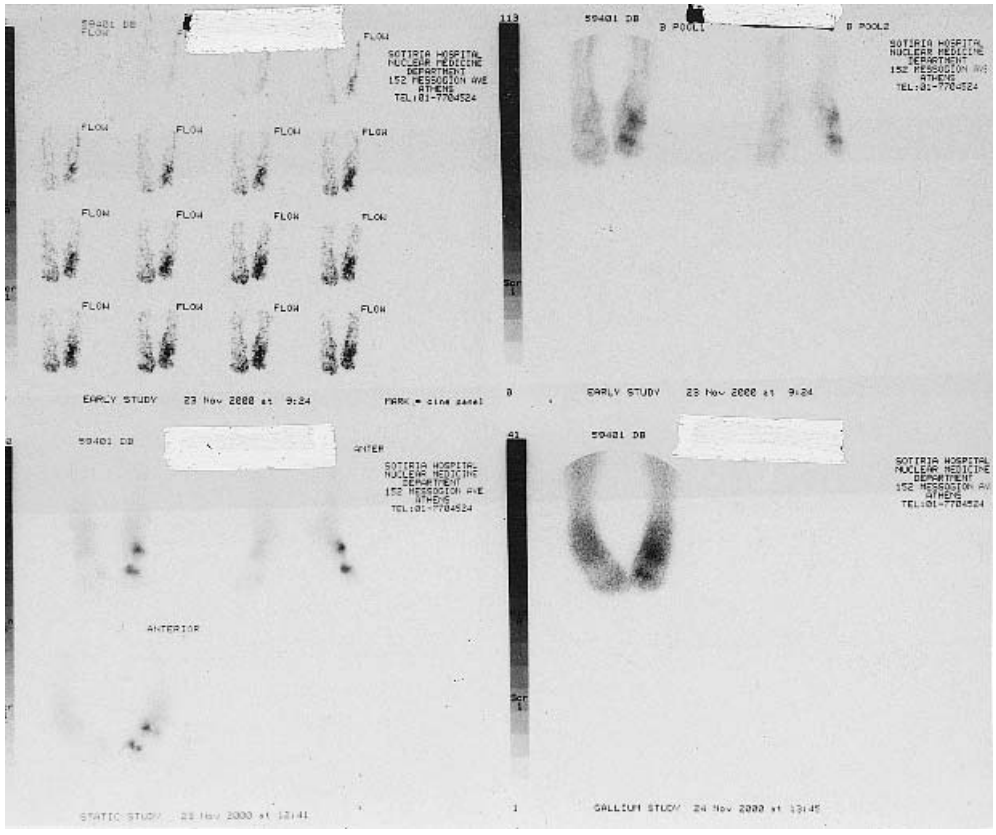

Figure 8.37 Increased radionuclide uptake by the tarsometatarsal bones, possibly due to osteomyelitis. Technetium-99m (^{99}Tc) phosphonates scan: *flow phase* (left upper panel); *blood pool phase* (right upper panel); *delayed phase* (left lower panel). Gallium-67 citrate study (right lower panel)

A plain radiograph showed a large skin defect on the posterioplantar aspect of her heel and bone resorption of the posterior calcaneus (Figure 8.39). Extensive calcinosis of the posterior tibial artery and medial plantar branch artery was also noted. After surgical debridement, bone and deep tissue cultures were obtained. Immediate support with i.v. fluids and antibiotic administration was commenced (ciprofloxacin 400 mg × 3 and clindamycin 600 mg × 3) and her situation improved within 12 h. Tissue cultures revealed *Enterococcus* spp., *Acinetobacter baumannii*, and *Proteus mirabilis*. Based on an antibiogram, treatment was changed to ampicillin–sulbactam and continued for 2 weeks. Disarticulation through the ankle joint (Syme ankle disarticulation) was not feasible; a healthy heel flap and the heel pad is a prerequisite for this procedure so that the end of the stump is capable of bearing the patient's weight. Two weeks after her admission the patient sustained a below-knee amputation.

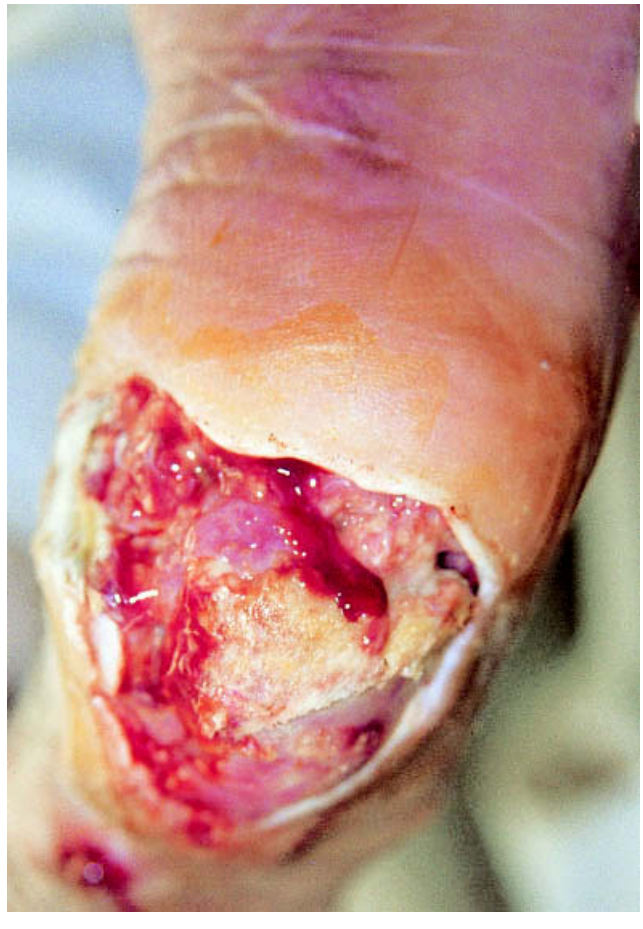

Figure 8.38 Large, neuro-ischemic ulcer with gross purulent discharge on the posterior surface of right heel. The calcaneus is exposed

Empirical treatment with antibiotics in severe foot infections should always include agents against staphylococci, enterobacteriaceae and anaerobes. In this case, two agents with good bone bioavailability were used since osteomyelitis was present. Therapeutic options in patients with severe foot infections include:

- Fluoroquinolone plus metronidazole or clindamycin. This combination is effective against *Staphylococcus aureus* (only methicillin-susceptible strains), enterobacteriaceae, and anaerobes.
- β-lactam and β-lactamase inhibitor combinations (ticarcilline–clavulanic acid, piperacillin–tazobactam). Ampicillin–sulbactam is particularly active against *Enterococcus* spp. For patients who have received extensive antibiotic therapy, ticarcilline–clavulanic acid or piperacillin–tazobactam may be preferred because of their increased activity against nosocomial gram-negative bacilli. Such regimens are also effective against

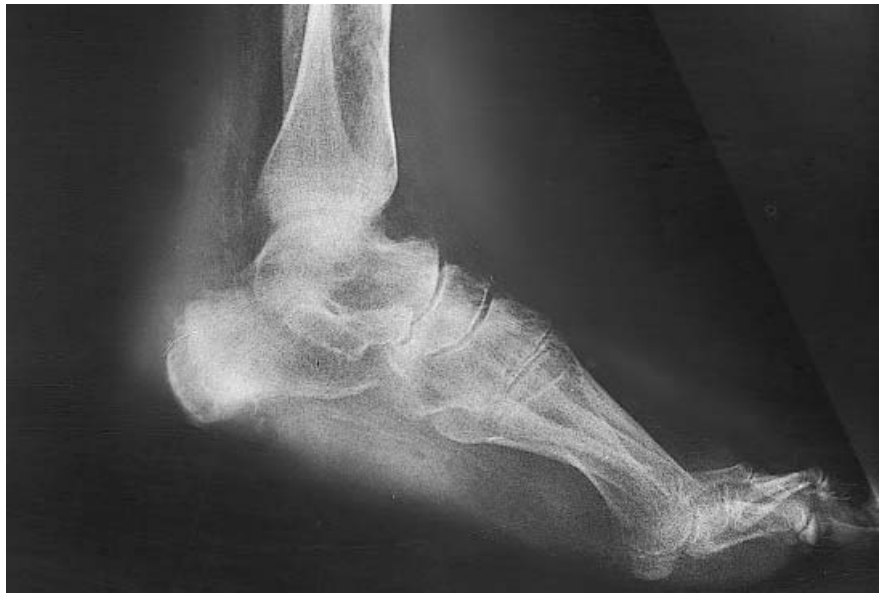

Figure 8.39 Plain radiograph of the foot illustrated in Figure 8.38 showing a large skin defect on the posterioplantar aspect of the heel and bone resorption of the posterior calcaneus. Calcinosis of the posterior tibial artery and medial plantar branch artery is also apparent

Staphylococcus aureus (only methicillin sodium-susceptible strains), *Streptococcus* spp. and most anaerobes.
- In patients who have severe penicillin allergy, combination therapy with aztreonam and clindamycin, or a fluoroquinolone and clindamycin is effective.
- Imipenem–cilastin or meropenem as monotherapy.

Doctors should always consider that:
- Modification of the treatment may be necessary according to the results of cultures.
- Vancomycin or teicoplanin are indicated in cases of infection with methicillin-resistant staphylococcal strains.
- Third generation cephalosporins should be used only in combination with other agents, as they have moderate anti-staphylococcal activity and lack significant activity against anaerobes.
- Aminoglycosides are nephrotoxic and they are inactivated in the acidic environment of the soft tissue infection and have poor penetration into bone.

Keywords: Osteomyelitis; heel ulceration; calcaneus; severe foot infection treatment; below-knee amputation

http://dratef.net/

http://dratefahmed.blogspot.com/

http://tv.dratef.net/

http://books.dratef.net/

http://feeds.feedburner.com/Dratefnet

https://www.facebook.com/onlinemedicalbook/

https://www.facebook.com/onlineSurgicalbook/

https://twitter.com/DrATEFAHMED

https://twitter.com/medicalbook1

https://www.instagram.com/dr.atefahmed/

https://www.linkedin.com/in/dratef/

https://www.pinterest.com/shoppingdealer2/

http://feeds.feedburner.com/semnatv/wxHK

http://shoppingdealer.com/

http://ymarryme.com/

http://semnatv.com/